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09/771,302	01/26/2001	Martha J. Whitehouse	1543.201 (5784-81A)	7656

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4560 Horton Street  
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EXAMINER

DEBERRY, REGINA M

ART UNIT	PAPER NUMBER
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1647

DATE MAILED: 05/07/2003

17

Please find below and/or attached an Office communication concerning this application or proceeding.

# Office Action Summary

Application No.

09/771,302

Applicant(s)

WHITEHOUSE, MARTHA J.

Examiner

Regina M. DeBerry

Art Unit

1647

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

## Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

## Status

- 1) ☒ Responsive to communication(s) filed on 10 March 2003.
- 2a) ☐ This action is FINAL. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

## Disposition of Claims

- 4) ☒ Claim(s) 35-52 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 35-52 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

## Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on \_\_\_\_\_ is: a) ☐ approved b) ☐ disapproved by the Examiner.
- If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

## Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
- a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

## Attachment(s)

- 1) ☐ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 3, 16.
- 4) ☐ Interview Summary (PTO-413) Paper No(s). \_\_\_\_\_.
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: \_\_\_\_\_.

Art Unit: 1647

***Status of Application, Amendments and/or Claims***

The amendment filed 11 February 2003 (Paper No. 15) has been entered in full.

The information disclosure statement filed 10 March 2003 (Paper No. 16) was received and complies with the provisions of 37 CFR §§1.97 and 1.98. It has been placed in the application file and the information referred to therein has been considered as to the merits.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

***Withdrawn Objections And/Or Rejections***

The rejections of claims 35, 36, 43 and 44 under 35 U.S.C. 103(a) as being unpatentable over Deisher *et al.*, US Patent No. 5,989,866 as set forth at pages 2-4 of the previous Office Action (15 November 2002, Paper No. 14) is *withdrawn*.

The rejections of claims 37 and 45 under 35 U.S.C. 103(a) as being unpatentable over Deisher *et al.*, US Patent No. 5,989,866 in view of Fiddes *et al.*, US Patent No. 5,604,293 as set forth at pages 4-5 of the previous Office Action (15 November 2002, Paper No. 14) is *withdrawn*.

The rejections of claims 38-41, 42, 47-49 under 35 U.S.C. 103(a) as being unpatentable over Deisher *et al.*, US Patent No. 5,989,866 in view of in view of Wilson *et al.*, US Patent No. 5,612,211 and Unger *et al.*, US Patent No. 5,244,460 as set forth at pages 5-6 of the previous Office Action (15 November 2002, Paper No. 14) is *withdrawn*.

**Claim Rejections - 35 USC § 103**

Claims 35, 36, 43 and 44 are rejected under 35 U.S.C. 103(a) as being unpatentable over Laham *et al.*, J. Am. Coll. Cardiol., March 1998 (IDS submitted by Applicant, #51) in view of Deisher *et al.*, US Patent No. 5,989,866.

Laham *et al.* teach that perivascular basic fibroblast growth factor (bFGF, FGF-2) delivery results in functionally significant angiogenesis in an animal model of chronic myocardial ischemia. Laham teaches the implantation of bFGF-heparin alginate microcapsules in the epicardial fat surrounding the vessel in the heart of human patients. Laham does not specifically teach the administration of bFGF via a coronary vessel or into a peripheral vein.

Deisher *et al.* teach polynucleotide and polypeptide molecules of zFGF-5, a novel member of the FGF family (abstract). Deisher teaches homology alignments with human FGF-2 (figures 1 and 2; column 5, lines 41-59; column 8, lines 20-45 and column 14, lines 37-62). The invention provides pharmaceutical compositions comprising a purified FGF homolog polypeptide, in combination with a pharmaceutically acceptable vehicle (column 4, lines 65-67). ZFGF-5 may be used in treatment of disorders associated with congestive heart failure (column 25, lines 30-34) and other indications where angiogenesis is of benefit (column 25, lines 35-46). Thus zFGF-5 can be considered an angiogenically active mutein or fragment thereof FGF-2. Deisher teaches intravenous administration of zFGF-5 by bolus injection or infusion. Doses are in the range of 0.1 to 100 ug/kg of patient weight per day, preferably 0.5-20ug/kg per day. Deisher also states that determination of dose is within the level of ordinary skill in

Art Unit: 1647

the art (column 26, lines 36-67). Deisher demonstrates that zFGF-5 has the *in vivo* activity of inducing cardiac mitogenesis (Examples 4 and 5).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to modify the teachings of Laham and Deisher to use the instant invention of a method of treating congestive heart failure comprising administering recombinant FGF-2 or an angiogenically active fragment or mutein thereof into a coronary vessel or peripheral vein in a human patient in need of treatment. The motivation and expected success is provided by both Laham and Deisher. Laham teaches that perivascular bFGF delivery results in functionally significant angiogenesis in an animal model of chronic myocardial ischemia and the successful implantation of bFGF-heparin alginate microcapsules in the heart of human patients. Deisher teaches that zFGF-5 has the *in vivo* activity of inducing cardiac mitogenesis. Deisher discloses the intravenous administration of zFGF-5 by bolus injection or infusion. Although the references do not teach administering a unit dose range of .008 mg to 7.2 mg to patients, these adjustments are a matter of judicial selection. Routine optimization is well within the purview of the skilled artisan.

Applicant states that FGF-2 and zFGF-5 are disclosed as being very different growth factors, both structurally and functionally. This is not found persuasive. Deisher teaches that zFGF-5 contains the CXFXE{6}Y motif present in all members of the FGF family (column 8, lines 20-25). Members of the FGF family are characterized by heparin binding domains. A putative heparin-binding domain for zFGF-5 has been identified (column 8, lines 57-60). Deisher states that "one skill in the art will recognize that other

Art Unit: 1647

members, in whole or part, of the FGF family may have structural or biochemical similarities to zFGF-5 (column 14, lines 57-60). Deisher discloses that zFGF-5 may also be useful for promoting angiogenesis and used in treatment of disorders associated with myocardial infarction, congestive heart failure and hypertrophic cardiomyopathy. (column 25, lines 30-38). Furthermore, the instant claims are drawn to "recombinant FGF-2 or an angiogenically active *fragment* or an angiogenically active *mutein thereof*", thus zFGF-5 can be considered a mutein or comprising a fragment thereof FGF-2.

Applicant also states that Deisher *et al.* do not teach or suggest a method for treating a human patient for congestive heart failure comprising administering an angiogenically active fragment or mutein of FGF-2 or provide evidence that zFGF-5 or its muteins have angiogenic activity or mitogenic activity for endothelial cells. Applicant notes the presence of clinical trials disclosed in the instant specification. Applicant states that the therapeutic benefits could not have been predicted or expected by those of skill in the art based on the teachings of Deisher *et al.* The features upon which Applicant relies upon, (mitogenesis, endothelial cells, therapeutic benefits recited in the instant specification), are not recited in the claims. Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

Art Unit: 1647

Claims 37 and 45 are rejected under 35 U.S.C. 103(a) as being unpatentable over Laham *et al.*, J. Am. Coll. Cardiol., and Deisher *et al.*, US Patent No. 5,989,866 in further in view of Fiddes *et al.*, US Patent No. 5,604,293.

The teachings of Laham and Deisher are described above. The Laham and Deisher references do not teach administration of recombinant FGF-2 with the amino acid sequence of SEQ ID NO:2. Fiddes teaches a recombinant FGF-2 protein comprising the sequence of SEQ ID NO:2 (Figure 4 and column 3, lines 48-49). Fiddes states that the invention provides the tools for synthesis and manipulation of fibroblast growth factors useful in effecting damaged myocardial tissue (column 3, lines 11-19). Fiddes teaches intravenous administration of FGF-2 (SEQ ID NO:2) (column 8, lines 30-41).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to modify the teachings of Laham and Deisher and Fiddes to use the instant invention of a method of treating congestive heart failure comprising administering recombinant FGF-2 (SEQ ID NO:2). The motivation and expected success is provided by Fiddes who teaches the activity of wound healing by subcutaneous implantation of bovine FGF-2 (SEQ ID NO:2) in rats. Fiddes also teaches that angiogenic stimuli, provided by FGF, results in the release of tissue plasminogen and collagenase and that the FGF proteins of the invention are also useful in treatment of conditions which respond to these enzymes (stroke, heart attack) (column 8, lines 25-41).

Art Unit: 1647

Applicant states that Fiddes *et al.* disclose the synthesis and manipulation of acidic (FGF-1) and basic (FGF-2) fibroblast growth factors and suggest that these sequences are useful in damaged myocardial tissue. Applicant asserts, that the only evidence of therapeutic utility for the claimed sequences resides in Example 12, where the potential for FGF-2 activity to promote wound healing is demonstrated by using subcutaneous implantation of bovine FGF-2 soaked polyvinyl alcohol sponges in rats. Applicant states that Fiddes *et al.* suggest intravenous administration of FGF-1 and FGF-2 for treatment of conditions that respond to tissue plasminogen activator or collagenase. Contrary to Applicant's assertion, Fiddes teaches that angiogenic stimuli, provided by FGF, results in the release of tissue plasminogen and collagenase and that the FGF proteins of the invention are also useful in treatment of conditions such as stroke and heart attack. The instant claims are drawn to a method for treating a patient for congestive heart failure. Congestive heart failure is caused by conditions such as myocardial infarction, stroke, and heart attack.

Applicant asserts that Fiddes *et al.* and Deisher *et al.* are silent with respect to administration of FGF-2 into coronary vessels or into a peripheral vein of a human patient in need of treatment for congestive heart failure. The Examiner recognizes that obviousness can only be established by combining or modifying the teachings of the prior art to produce the claimed invention where there is some teaching, suggestion, or motivation to do so found either in the references themselves or in the knowledge generally available to one of ordinary skill in the art. See *In re Fine*, 837 F.2d 1071, 5 USPQ2d 1596 (Fed. Cir. 1988) and *In re Jones*, 958 F.2d 347, 21 USPQ2d 1941 (Fed.



Art Unit: 1647

Cir. 1992). In this case, Laham teaches that perivascular bFGF delivery results in functionally significant angiogenesis in an animal model of chronic myocardial ischemia and the successful implantation of bFGF-heparin alginate microcapsules in the heart of human patients. Deisher discloses that FGF-2 has been shown to play a role in avian cardiac development and that FGF-2 and zFGF-5 have both been shown to induce coronary collateral development in animal models. Deisher discloses that zFGF-5 can be formulated for intravenous administration. Deisher teaches that zFGF-5 has mitogenic activity. Fiddes discloses that FGF is useful in damaged myocardial tissue and in the treatment of conditions, which respond to these enzymes (stroke, heart attack). Fiddes teaches the same sequence recited in the instant claims (SEQ ID NO:2) and that this sequence promotes wound healing in rats.

Applicant asserts that one of skill in the art would recognize that such limited observations in avian and canine models are not readily applicable to humans for reasons noted above and further elaborated on in the present specification (at page 3). This is not found to be persuasive. The instant specification cites Battler who asserts that caution must be exercised in extrapolating results from different animal models (specification, page 3, lines 17-21). This statement, however, does not discourage the use of animal models. Furthermore, Laham teaches the implantation of bFGF-heparin microcapsules in the heart of human patients. One skilled in the art would be motivated to use FGF-2 to treat congestive heart failure because the literature does not teach against it.

Art Unit: 1647

Claims 38-41, 42, 47-49 are rejected under 35 U.S.C. 103(a) as being unpatentable over Laham *et al.*, J. Am. Coll. Cardiol., in view of Deisher *et al.*, US Patent No. 5,989,866, Fiddes *et al.*, US Patent No. 5,604,293, Wilson *et al.*, US Patent No. 5,612,211 and Unger *et al.*, US Patent No. 5,244,460.

The teachings are Laham, Deisher and Fiddes are described above. The Laham, Deisher and Fiddes references do not teach the administration of heparin and FGF via a coronary vessel or peripheral vein.

Wilson teaches that heparin may be used according to the present invention to potentiate the stimulatory effect of concentrations of an FGF (column 12, line 52-63). A therapeutically effective amount of at least one FGF optionally in combination with a therapeutically effective amount of at least one CSF and /or heparin may be administered by various routes including intravenously (column 14, lines 42-50). Wilson teaches administration of FGF dosage range of .02 ug/kg-2.0 mg/kg body weight or any range therein (column 15, lines 39-54). Wilson states that the methods of the present invention contemplates the use of at least one CSF and/or heparin, such as 1,2,3,4,5,6,7,8,9 or 10 heparins and/or CSFs or any range therein in combination with at least one FGF (column 16, lines 36-43). Although, Wilson does not teach exact heparin concentrations or administration of heparin within 30 minutes of administering recombinant FGF-2 of SEQ ID NO:2 or said angiogenically active fragment or said angiogenically active mutein thereof, one skilled in the art would have been motivated to modify Wilson *et al.* to include the adjustments of conventional working conditions such as concentrations and times points of administration.

Art Unit: 1647

Unger teaches the administration of drugs into the coronary artery (abstract, and column 4, lines 18-52). Unger states that there is a need to target agents directly to the heart in order to promote the growth of new cardiac blood vessels and a need for the method to treat patients suffering with atherosclerosis of the coronary arteries (column 3, lines 58-67). Unger teaches the administration of bFGF (FGF-2) in amounts between 50-700 ug into the coronary artery (column 6, lines 32-38; column 7, lines 54-67 and column 8, lines 15-67).

It would have been obvious to one of ordinary skill in the art at the time the invention was made to modify the invention of a method to treat congestive heart failure using the heparin teachings disclosed by Wilson and coronary artery teachings disclosed by Unger. The motivation and expected success is provided by Wilson, who teaches that heparin potentiates the stimulatory effect of FGF and Unger who teaches methods comprising administering into a coronary artery, bFGF (FGF-2) for treating a damaged heart.

Applicant maintains that Wilson *et al.* is directed to FGF stimulation of stem cells, either *in vivo*, or *in vitro* followed by transplantation or engraftment, for purposes of treating a long list of diseases or pathologies, though no mention is made of patients with congestive heart failure. Applicant asserts that the reference teaches the use of heparin to potentiate the stimulatory effects of concentrations of an FGF administered to a hematopoietic cell donor, recipient or subject according to a method of the present invention. Applicant contends that the motivation to combine the teachings of Wilson *et al.* with the teachings of Deisher *et al.* is lacking. Applicant maintains that even if one of

Art Unit: 1647

skill in the art were motivated to modify the invention of Deisher *et al.* to include administration of heparin based on the disclosure of Wilson *et al.*, such a modification would not produce Applicant's claimed invention.

This is not found to be persuasive. It has been held that a prior art reference must either be in the field of applicant's endeavor or, if not, then be reasonably pertinent to the particular problem with which the applicant was concerned, in order to be relied upon as a basis for rejection of the claimed invention. See *In re Oetiker*, 977 F.2d 1443, 24 USPQ2d 1443 (Fed. Cir. 1992). In this case, Wilson teaches that FGFs are expected to increased proliferation, stimulation, growth and/or differentiation of blood cells (column 11, lines 52-59). Wilson discloses that heparan sulfate is known to be a component of the extracellular matrix which influences hemopoiesis and that growth factors have been shown to bind heparin, serving as a possible mechanism by which stromal cells affect hemopoiesis (column 12, lines 45-50). Hemopoiesis is the formation and development of blood cells. Deisher (above) teaches that members of the FGF family are characterized by heparin binding domains. It would be obvious to use heparin with FGF-2 in a method for treating a patient with congestive heart failure. FGF-2 has been implicated in angiogenesis and heparin will not only bind and potentiate the stimulatory effects of the concentration of FGF but also influence hemopoiesis. Hemopoiesis would be needed in functions involving neovascularization.

Applicant notes that the statements in Wilson *et al.* are irrelevant to the obvious rejection. Wilson disclosed that FGF was used in the treatment of ischemic heart disease (Franco, US Patent Nos. 4,296,100 and 4,378,347) and might induce

Art Unit: 1647

neovascularization (Arakawa *et al.*, EP 320148). Applicant contends that the Franco patents fail to demonstrate safety and/or efficacy in either animal model disclosed. Applicant notes that models of acute myocardial infarction are not predictive for a method of treatment for congestive heart failure, which represents a chronic ischemic condition as opposed to an acute ischemic event. Applicant states that the Arakawa *et al.* reference shows the ideal that FGF-2 might induce neovascularization was from an *in vitro* assay. Applicant states that the findings of Franco and Arakawa *et al.* cannot be construed as providing any reasonable expectation of success in using FGF for treating a human patient for congestive heart failure.

This is not found persuasive. The instant rejections were based on the combined references of Laham, Deisher, Fiddes, Wilson and Unger, which when combined teach all of the limitations of the instant claims. Furthermore, the specification does not specifically define congestive heart failure as a chronic ischemic condition as opposed to an acute ischemic event. Laham teaches that perivascular bFGF delivery results in functionally significant angiogenesis (neovascularization) in an animal model of chronic myocardial ischemia.

Applicant assert that the Unger *et al.* reference requires repeated injections until improved cardiac blood flow has been obtained. Applicant states that in contrast, the current invention provides a method comprising administering a single unit dose of FGF-2 for coronary angiogenesis. Applicant states that the Unger reference teaches away from administration of a single unit dose of FGF-2 for congestive heart failure. This is not found persuasive as independent claim 35 does not have the limitation "a

Art Unit: 1647

single unit dose". Furthermore, the Unger *et al.* reference would not only teach against the Franco reference but against instant claims 43-52.

**Double Patenting**

Claims 35-52 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-58 of U.S. Patent No. 6,440,934 B1. Although the conflicting claims are not identical, they are not patentably distinct from each other. The instant claims are drawn to a method for treating a human patient for congestive heart failure comprising administering a therapeutically effective amount of a recombinant FGF-2 or an angiogenically active fragment or an angiogenically active mutein thereof into one or more coronary vessels or into a peripheral vein in a human patient in need thereof of treatment for said congestive heart failure, said therapeutically effective amount being about 0.2µg/kg to 48µg/kg of patient weight. The instant claims are also drawn to a method for treating a human patient for congestive heart failure comprising administering a single unit dose of a recombinant FGF-2 or an angiogenically active fragment or an angiogenically active mutein thereof into one or more coronary vessels or into a peripheral vein in a human patient in need thereof of treatment for congestive heart failure, said unit dose comprising from about .008mg to 7.2mg of said recombinant FGF-2 or said angiogenically active fragment or said angiogenically active mutein thereof.

The claims of U.S. Patent No. 6,440,934 B1 are drawn to a method for treating a human patient for coronary artery disease, a method for inducing angiogenesis, a

Art Unit: 1647

method for treating a human patient for a myocardial infarction and a method for providing a human patient with relief from symptoms of angina.

The instant claims and the claims of patent '934 are both drawn to administering the same amounts of recombinant FGF-2 or an angiogenically active fragment or an angiogenically active mutein thereof into one or more coronary vessels or into a peripheral vein in a human patient. The instant claims and the claims of patent '934 are both drawn to administering the same amounts heparin at the same time. The instant claims and the claims of patent '934 are both drawn to the same therapeutic benefits of administering recombinant FGF-2 or an angiogenically active fragment or an angiogenically active mutein thereof. The instant claims and the claims of patent '934 are both drawn to administering a single unit dose of a recombinant FGF-2 or an angiogenically active fragment or an angiogenically active mutein thereof into one or more coronary vessels or into a peripheral vein in a human patient. The instant claims and the claims of patent '934 are both drawn to administration into coronary arteries, vessels or peripheral veins.

The claims of patent '934 are not drawn to a method of treating congestive heart failure, however, congestive heart failure is a condition where there is ineffective pumping of the heart leading to an accumulation of fluid in the lungs. This term encompasses the diseases recited in patent '934 (coronary artery disease and myocardial infarction). Angina is chest pain. Chest pain would occur in congestive heart failure. The instant claims are drawn to angiogenically active fragments of FGF-2, thus those fragments when administered would cause angiogenesis. The claims of patent

Art Unit: 1647

'934 are already drawn to a method for inducing angiogenesis. It would have been obvious to one of ordinary skill in the art to design the instant method for treatment of congestive heart failure comprising administering recombinant FGF-2 or an angiogenically active fragment or an angiogenically active mutein thereof for since such methods are encompassed by the invention of patent '934 which teaches treatments for diseases or conditions which fall under congestive heart failure. The species renders the genus obvious.

***Conclusion***

No claims are allowed.



Art Unit: 1647

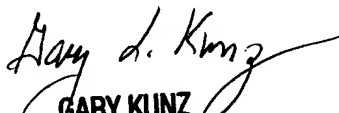
Any inquiry concerning this communication or earlier communications from the examiner should be directed to Regina M. DeBerry whose telephone number is (703) 305-6915. The examiner can normally be reached on 9:00 a.m.-6:00 p.m..

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Kunz can be reached on (703) 308-4623. The fax phone numbers for the organization where this application or proceeding is assigned are (703) 872-9306 for regular communications and (703) 872-9307 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is (703) 308-0196.



RMD  
May 2, 2003



GARY KUNZ  
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